Amendments to the Claims

This listing of claims is intended to replace all prior versions, and listings, of claims presented in the above-identified application:

1. (currently amended) A method of treating pathogen infection in a subject, said method comprising:

inhibiting proteasomal activity in a pathogen ~~under conditions effective~~ to make the pathogen susceptible to antibacterial host defenses, thereby treating a pathogen infection in the subject.

2. (original) The method according to claim 1, wherein the proteasomal activity is an ~~AAA~~ ATPase activity or a proteasomal protease activity.

3. (original) The method according to claim 2, wherein the proteasomal activity is proteasomal protease activity in a proteasome core.

4. (currently amended) The method according to claim 3, wherein the proteasomal protease is a product of *prcBA* genes.

5. (original) The method according to claim 4, wherein the protease is PrcA.

6. (original) The method according to claim 4, wherein the protease is PrcB.

7. (withdrawn) The method according to claim 2, wherein the proteasomal activity is an AAA ATPase activity where the AAA ATPase is selected from the group consisting of an AAA ATPase forming ring-shaped complex, a proteasome associated nucleotidase, a mycobacterial proteasome ATPase, and a proteasome accessory factor.

8. (withdrawn) The method according to claim 7, where the AAA ATPase is an AAA ATPase forming ring-shaped complex.

9. (withdrawn) The method according to claim 8, wherein AAA ATPase forming ring-shaped complex is a product of a groEL1 gene.

10. (withdrawn) The method according to claim 9, where the groEL gene product is a groEL protein.

11. (withdrawn) The method according to claim 8, wherein the AAA ATPase forming ring-shaped complex is a product of a Rv2115c gene.

12. (original) The method according to claim 1, wherein the host defense is oxidative/nitrosative stress.

13. (original) The method according to claim 12, wherein the oxidative/nitrosative stress is reactive nitrogen intermediate-induced stress.

14. (original) The method according to claim 12, wherein the oxidative/nitrosative stress is reactive oxygen intermediate-induced stress.

15. (original) The method according to claim 1, wherein the inhibiting is carried out by administering an inhibitor of proteasomal activity.

16. (original) The method according to claim 15, wherein the administering is oral, intradermal, intramuscular, intraperitoneal, intravenous, subcutaneous, or intranasal.

17. (original) The method according to claim 15, wherein the inhibitor of proteasomal activity is selected from the group consisting of epoxomicin and N-[4-morpholine]carbonyl-β-[1-naphthyl]-L-alanine-L-leucine boronic acid.

18. (original) The method according to claim 1, wherein the pathogen is selected from the group consisting of *Mycobacterium tuberculosis*, *Mycobacterium leprae*, and another disease-causing *Mycobacterium*.

19. (original) The method according to claim 1, wherein the subject is a mammal.

20. (original) The method according to claim 19, wherein the mammal is human.

21-62. (canceled)

63. (currently amended) A method of treating pathogen infection in a subject, said method comprising:

(1) inhibiting proteasomal activity in a pathogen ~~under conditions effective~~ to make the pathogen susceptible to antibacterial host defenses and

(2) inhibiting enzyme activity in the pathogen ~~under conditions effective~~ to make the pathogen susceptible to antibacterial host defenses, thereby treating the pathogen infection in a host.

64. (currently amended) The method according to claim 63, wherein the proteosomal activity is an AAA ATPase activity or a proteasomal protease activity.

65. (currently amended) The method according to claim 64, wherein the proteasomal activity is proteasomal protease activity where a proteasomal core is being inhibited.

66. (original) The method according to claim 65, wherein the protease is a product of the *prcBA* gene.

67. (original) The method according to claim 66, wherein the protease is PrcB.

68. (original) The method according to claim 66, wherein the protease is PrcA.

69. (withdrawn) The method according to claim 64, wherein the proteasomal activity is an AAA ATPase activity, where the AAA ATPase is selected from

the group consisting of an AAA ATPase forming ring-shaped complex, a proteasome associated nucleotidase, a mycobacterial proteasome ATPase, and a proteasome accessory factor.

70. (withdrawn) The method according to claim 69, wherein the AAA ATPase is an AAA ATPase forming ring-shaped complex.

71. (withdrawn) The method according to claim 70, wherein the AAA ATPase forming ring-shaped complex is a product of a groEL1 gene.

72. (withdrawn) The method according to claim 71, where the groEL gene product is a groEL protein.

73. (withdrawn) The method according to claim 70, wherein the AAA ATPase forming ring-shaped complex is a product of an Rv2115c gene.

74. (original) The method according to claim 63, wherein the inhibiting is carried out by administering an inhibitor of proteasomal activity.

75. (original) The method according to claim 74, wherein the inhibitor of proteasomal activity is selected from the group consisting of epoxomicin and N-[4-morpholine]carbonyl-β-[1-naphthyl]-L-alanine-L-leucine boronic acid.

76. (original) The method according to claim 74, wherein the administering is oral, intradermal, intramuscular, intraperitoneal, intravenous, subcutaneous, or intranasal.

77. (currently amended) The method according to claim 63, wherein the enzyme is selected from the group consisting of a DNA repair enzyme and a ~~flavin-like co-factor~~ co-enzyme F420 biosynthesis enzyme.

78. (currently amended) The method according to claim 77, wherein the enzyme inhibited is a DNA repair enzyme in the form of a ~~nucleotidase~~ nucleotide excision-repair enzyme.

79. (currently amended) The method according to claim 78, wherein the ~~nucleotidase~~ nucleotide excision-repair enzyme is a product of a *uvr* gene ~~family~~.

80. (currently amended) The method according to claim 79, wherein the ~~nucleotidase~~ nucleotide excision-repair enzyme is UvrB.

81. (currently amended) The method according to claim 77, wherein the enzyme inhibited is a ~~flavin-like co-factor~~ co-enzyme F420 biosynthesis enzyme.

82. (original) The method according to claim 63, wherein the host defense is oxidative/nitrosative stress.

83. (original) The method according to claim 82, wherein the oxidative/nitrosative stress is reactive nitrogen intermediate-induced stress.

84. (original) The method according to claim 82, wherein the oxidative/nitrosative stress is reactive oxygen intermediate-induced stress.

85. (original) The method according to claim 63, wherein the pathogen is selected from the group consisting of *Mycobacterium tuberculosis*, *Mycobacterium leprae*, and another disease-causing *Mycobacterium*.

86. (original) The method according to claim 63, wherein the subject is a mammal.

87. (original) The method according to claim 86, wherein the mammal is human.

88-102 (canceled)